

$\Rightarrow P$ 

E25	3	GLOMERULONEPHROSIS/BI
E26	3	GLOMERULONEPHRITIS/BI
E27	3	GLOMERULONEPHRITIS/BI
E28	19	GLOMERULOPATHIES/BI
E29	11	GLOMERULOPATHY
E30	1	GLOMERULOPHOSIS/BI
E31	1	GLOMERULOPHOSIS/BI
E32	63	GLOMERULOSA/BI
E33	10	GLOMERULOSAL/BI
E34	114	GLOMERULOSCLEROSIS/BI
E35	1	GLOMERULOSCLEROSIS/BI
E36	2	GLOMERULOSCLEROTIC/BI

=> s e8-e29

1 GLOMERULONEPHRITIS/BI  
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 4 GLOMERULONEPHRITIS/BI  
 1 GLOMERULONEPHRAL/BI  
 1 GLOMERULONEPHRIS/BI  
 1 GLOMERULONEPHRISIS/BI  
 9 GLOMERULONEPHRITES/BI  
 6 GLOMERULONEPHRITIC/BI  
 7 GLOMERULONEPHRITIDES/BI  
 1074 GLOMERULONEPHRITIS/BI  
 1 GLOMERULONEPHRITISO/BI  
 1 GLOMERULONEPHRITITS/BI  
 3 GLOMERULONEPHRITUS/BI  
 3 GLOMERULONEPHROPATHY/BI  
 3 GLOMERULONEPHROSIS/BI  
 3 GLOMERULONEPHRITRITIS/BI  
 1 GLOMERULONEPHTRITIS/BI  
 19 GLOMERULOPATHIES/BI  
 13 GLOMERULOPATHY/BI  
 L10 1114 (GLOMERULONEPHRITIS/BI OR GLOMERULONEPHRITIS/BI OR  
 GLOMERULONEPHRITIS/BI OR GLOMERULONEPHRITIS/BI OR  
 GLOMERULONEPHRAL/BI OR GLOMERULONEPHRIS/BI OR GLOMERULONEPHRISIS/BI OR  
 GLOMERULONEPHRITES/BI OR GLOMERULONEPHRITIC/BI OR  
 GLOMERULONEPHRITID  
 ES/BI OR GLOMERULONEPHRITIS/BI OR  
 GLOMERULONEPHRITISO/BI  
 OR GLOMERULONEPHRITITS/BI OR GLOMERULONEPHRITUS/BI  
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 OR GLOMERULONEPHROPATHY/BI OR GLOMERULONEPHROSIS/BI  
 GLOMERULONEPHRITRITIS/BI OR GLOMERULONEPHRITRITIS/BI OR  
 GLOMERULOPATHIES/BI OR GLOMERULOPATHY/BI)

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(FILE 'HOME' ENTERED AT 17:13:09 ON 15 AUG 1998)

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L1      FILE 'USPATFULL' ENTERED AT 17:14:49 ON 15 AUG 1998
L2      380 S CHRONIC RENAL FAILURE
L2      18112 S ANTIINFLAMM? OR (ANTI INFLAMMAT?) OR
IMMUNOSUPPRESS?
L3      78 S L1 AND L2
L4      845 S (BONE(W)MORPHOGEN?) OR BMP? OR
(OSTEOGENIC(W)PROTEIN?)
L5      1271 S TGFEBETA## OR (TGF(W)BETA##) OR
{(TRANSFORMING(W)GROWTH
L6      26 S L2(P)L4
L7      109 S L2(P)L5
L8      0 S L1 AND L6
L9      0 S L1 AND L7
E GLOMERULO?
L10     1114 S EB-E29

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=> s 110 and 16

L11                    0 L10 AND L6

=> s 110 and 17

L12 15 L10 AND L7

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=> select
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ENTER ANSWER SET OR SMARTSELECT L# OR (L12):112

ENTER ANSWER NUMBER OR RANGE (1-): 1-15

ENTER DISPLAY CODE (TI) OR 2:nn

E1 THROUGH E15 ASSIGNED

=> s e1-e15

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1 US5061786/PN
1 US5268455/PN
1 US5605930/PN
1 US5635532/PN
1 US5635533/PN
1 US5654333/PN
1 US5661179/PN
1 US5688765/PN
1 US5693607/PN
1 US5693610/PN
1 US5708025/PN
1 US5710178/PN
1 US5712307/PN
1 US5747532/PN
1 US5759836/PN
L13 15 (US5061786/PN OR US5268455/PN OR US5605930/PN OR
US5635532 /PN OR US5635533/PN OR US5654333/PN OR
US5661179/PN OR US5688765/PN OR US5693607/PN OR US5693610/PN OR
US5708025/

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(FILE 'HOME' ENTERED AT 17:13:09 ON 15 AUG 1998)

L13 15 US5755838/FN  
US5635532 (US5061786/PN OR US5268455/PN OR US5605930/PN OR  
/PN OR US5635533/PN OR US5654333/PN OR  
US5661179/PN OR US5688765/PN OR US5693607/PN OR US5693610/PN OR  
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OR PN OR US5710178/PN OR US5712307/PN OR US5747532/PN  
US5759836/PN)

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(FILE 'HOME' ENTERED AT 17:13:09 ON 15 AUG 1998)

FILE 'USPATFULL' ENTERED AT 17:14:49 ON 15 AUG 1998

L1 380 S CHRONIC RENAL FAILURE  
L2 18112 S ANTIINFLAMM? OR (ANTI INFLAMMAT?) OR  
IMMUNOSUPPRESS?  
L3 78 S L1 AND L2  
L4 845 S (BONE(W)MORPHOGEN?) OR BMP? OR  
(OSTEOGENIC(W)PROTEIN?)  
L5 1271 S TGFbeta## OR (TGF(W)BETA##) OR  
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L6 26 S L2(P)L4  
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L8 0 S L1 AND L6  
L9 0 S L1 AND L7  
E GLOMERULO?  
L10 1114 S E8-E29  
L11 0 S L10 AND L6  
L12 15 S L10 AND L7  
SELECT L12 1-15 PN  
L13 15 S E1-E15

=> s l13 and l7

L14 15 L13 AND L7

=> d kwic 1-15

L14 ANSWER 1 OF 15 USPATFULL  
PI US 5759836 980602

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SUMM . . . Pharmacol. 216, 379-383, 1992). This is further  
substantiated by the observation that the upregulation of  
iNOS can be reduced by \*\*\*anti\*\*\* - \*\*\*inflammatory\*\*\*  
cytokines such  
as IL-4, IL-8, IL-10, \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .-1,  
-2, and  
-3, and macrophage deactivating factor (Nussler and  
Billiar, 1993,  
supra). COX-2 is induced in a number of cell. . . (Lin  
et al.,  
J. Biol. Chem. 264:17379-17383, 1989), IL-1 (Raz et al.,  
Proc.  
Natl. Acad. Sci. USA 86:1657-1661, 1989) and \*\*\*TGF\*\*\*  
-.  
\*\*\*beta\*\*\* . (Bailey and Verma, Anal. Biochem. 196:11-18,  
1991).  
PGE.sub.2 inhibits the production of cytokines (Ferreri et  
al., J.  
Biol. Chem. 267:9443-9449, . . .

L14 ANSWER 2 OF 15 USPATFULL  
PI US 5747532 980505

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DETD . . . protein, bactericidal/permeability increasing  
protein,  
polymyxin B, and the like), inhibition of cytokine  
synthesis/release (e.g., employing phosphodiesterase  
inhibitors,  
IL-4, IL-10, IL-13, \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .,  
corticosteroids, and the like), anti-cytokine therapy  
(e.g.,  
employing antibodies to TNF, soluble TNF receptors, IL-1  
receptor  
antagonists, antibodies to IL-1. . . and the like),  
inhibition  
of nitric oxide synthase enzymes (e.g., employing  
N-methyl-L-arginine, .epsilonpsilon-N-iminoethyl-L-lysine,  
aminoguanidine, S-methyl isothiourea sulfate, and the  
like),  
\*\*\*immunosuppression\*\*\* (e.g., employing agents such as  
cyclosporin A, OKT3, FK506, and the like), diabetic  
therapy (e.g.,  
employing agents such as free. . .

L14 ANSWER 3 OF 15 USPATFULL  
PI US 5712307 980127

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DETD . . . profound changes in tumor behavior were  
accompanied by  
alterations in the expression of genes implicated in  
growth  
control, angiogenesis, and \*\*\*immunosuppression\*\*\*  
(e.g.,  
TGF.alpha., HbF, and \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .2).  
DETD . . . athymic mice. Molecular analysis of brain and  
hormone-refractory prostate cancer cells revealed marked  
decline  
in the production and secretion of \*\*\*TGF\*\*\* .  
\*\*\*beta\*\*\* .,  
a protein implicated in growth control, angiogenesis, and  
\*\*\*immunosuppression\*\*\* . Treated prostatic cells  
exhibited  
decreased proteolytic activity mediated by  
urokinase-plasminogen  
activator, a molecular marker of disease progression in  
man.  
DETD The malignant prostatic cell lines exhibit numerous  
abnormalities  
in gene expression, including increased production of  
autocrine  
tumor growth factor-.beta. ( \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\*  
) and  
elevated activity of urokinase plasminogen activator  
(uPA).  
Members of the \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . family have  
been  
implicated in tumor growth control, angiogenesis, and  
\*\*\*immunosuppression\*\*\* . uPA, in contrast, is a serine  
protease  
involved in degradation of extracellular stroma and basal  
lamina

structures, with the potential to facilitate tumor  
invasion and  
metastasis. It was of interest, therefore, to examine the  
effect  
of NaPA on \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . and uPA  
expression in  
the prostatic tumor cells. Northern blot analysis of PC3  
after 72  
h treatment revealed a decrease in \*\*\*TGF\*\*\* -.  
\*\*\*beta\*\*\* .2  
mRNA levels; the effect was specific for \*\*\*TGF\*\*\* -.  
\*\*\*beta\*\*\* .2 as there was no change in the expression of  
\*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .1. The decrease in \*\*\*TGF\*\*\*  
-.  
\*\*\*beta\*\*\* .2 was accompanied by approximately a twofold  
increase  
in the levels of HLA-A3 mRNA, as previously observed in  
treated  
human leukemic. . .

L14 ANSWER 4 OF 15 USPATFULL  
PI US 5710178 980120

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DETD . . . profound changes in tumor behavior were  
accompanied by  
alterations in the expression of genes implicated in  
growth  
control, angiogenesis, and \*\*\*immunosuppression\*\*\*  
(e.g.,  
TGF.alpha., HbF, and \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .2).  
DETD . . . athymic mice. Molecular analysis of brain and  
hormone-refractory prostate cancer cells revealed marked  
decline  
in the production and secretion of \*\*\*TGF\*\*\* .  
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activator, a molecular marker of disease progression in  
man.  
DETD The malignant prostatic cell lines exhibit numerous  
abnormalities  
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) and  
elevated activity of urokinase plasminogen activator  
(uPA).  
Members of the \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . family have  
been  
implicated in tumor growth control, angiogenesis, and  
\*\*\*immunosuppression\*\*\* . uPA, in contrast, is a serine  
protease  
involved in degradation of extracellular stroma and basal  
lamina  
structures, with the potential to facilitate tumor  
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effect  
of NaPA on \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . and uPA  
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the prostatic tumor cells. Northern blot analysis of PC3  
after 72  
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\*\*\*beta\*\*\* .2  
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-.  
\*\*\*beta\*\*\* .2 was accompanied by approximately a twofold  
increase  
in the levels of HLA-A3 mRNA, as previously observed in  
treated  
human leukemic. . .

L14 ANSWER 5 OF 15 USPATFULL  
PI US 5708025 980113

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DETD . . . profound changes in tumor behavior were  
accompanied by  
alterations in the expression of genes implicated in  
growth  
control, angiogenesis, and \*\*\*immunosuppression\*\*\*  
(e.g.,  
TGF.alpha., HbF, and \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .2).  
DETD . . . athymic mice. Molecular analysis of brain and  
hormone-refractory prostate cancer cells revealed marked  
decline  
in the production and secretion of \*\*\*TGF\*\*\* .  
\*\*\*beta\*\*\* .,  
a protein implicated in growth control, angiogenesis, and  
\*\*\*immunosuppression\*\*\* . Treated prostatic cells  
exhibited  
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urokinase-plasminogen  
activator, a molecular marker of disease progression in  
man.  
DETD The malignant prostatic cell lines exhibit numerous  
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tumor growth factor-.beta. ( \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\*  
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structures, with the potential to facilitate tumor  
invasion and  
metastasis. It was of interest, therefore, to examine the  
effect  
of NaPA on \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . and uPA  
expression in  
the prostatic tumor cells. Northern blot analysis of PC3

after 72 h treatment revealed a decrease in \*\*\*TGF\*\*\* -.  
\*\*\*beta\*\*\* .2 mRNA levels; the effect was specific for \*\*\*TGF\*\*\* -.  
\*\*\*beta\*\*\* .2 as there was no change in the expression of  
\*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .1. The decrease in \*\*\*TGF\*\*\*  
-.  
\*\*\*beta\*\*\* .2 was accompanied by approximately a twofold  
increase in the levels of HLA-A3 mRNA, as previously observed in  
treated human leukemic. . .  
L14 ANSWER 6 OF 15 USPATFULL  
PI US 5693610 971202  
SUMM \*\*\*Transforming\*\*\* \*\*\*growth\*\*\* \*\*\*factor\*\*\* -.  
\*\*\*beta\*\*\* . ( \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .) and  
platelet-derived growth factor (PDGF) belong to such a  
growth factor. \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . was first found as  
a factor to promote growth of a rat fibroblast. Thereafter, it has  
been found that \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . inhibits growth  
of a lot of cells, strongly suppresses immunological activity, and  
increases extracellular matrix. It is suggested that the  
overproduction and/or abnormal metabolism of \*\*\*TGF\*\*\*  
-.  
\*\*\*beta\*\*\* . are involved in various diseases and  
symptoms of \*\*\*immunosuppression\*\*\* in a cancer patient or the like,  
fibroid lung, hepatic fibrosis, glomerulonephritis, scleroderma,  
or the like. Further, PDGF acts on. . .  
L14 ANSWER 7 OF 15 USPATFULL  
PI US 5693607 971202  
SUMM This invention relates to the fields of drug therapy and  
protein synthesis. A soluble \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .  
binding protein fragment is used to treat conditions characterized  
by an excess of \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* ., including  
present fibroproliferation and \*\*\*immunosuppression\*\*\*. The  
binding invention also relates to recombinant expression of the  
SUMM protein fragment in prokaryotic and eukaryotic cells.  
of There have been several attempts to suppress the effects  
antibody of \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . excess by administering  
pending which is specific for \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . In a  
patent application Ser. No. 759,109, filed Sep. 6, 1991,  
now U.S. Pat. No. 5,571,714 also assigned to Celtrix  
Pharmaceuticals, Inc., monoclonal antibodies to \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .  
were shown to have affinity constants ranging from 1.6.times.10.sup.7  
L/mol to 3.4.times.10.sup.8 L/mol in a competitive  
radioimmunoassay test. These monoclonal antibodies were suggested for use  
in treating tumor cells that produce \*\*\*TGF\*\*\* -.  
\*\*\*beta\*\*\* . to counteract the \*\*\*immunosuppressive\*\*\* effects of  
\*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . Another proposed use was  
treating metastatic cancers.  
SUMM In another embodiment of the present invention, the method  
provides for administration of \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .  
receptor fragment in the condition of \*\*\*TGF\*\*\* -.  
\*\*\*beta\*\*\* . excess characterized by \*\*\*immunosuppression\*\*\*  
associated with an infectious disease. In a further embodiment, the  
\*\*\*immunosuppression\*\*\* may be associated with  
trypanosomal infection or viral infections such as human  
virus \*\*\*immunosuppression\*\*\* virus, human T cell lymphotropic  
(HTLV-1), lymphocytic choriomeningitis virus and  
hepatitis. A sufficient amount of \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .  
DRWD -binding receptor fragment" as used herein refers to the amount of  
neutralizes the biologic activity of excess \*\*\*TGF\*\*\* -.  
\*\*\*beta\*\*\* . It may be determined by (1) suitable clinical variables of  
improvement, (2) pathologic evaluation of the effects on  
fibrosis and/or \*\*\*immunosuppression\*\*\* or prevention of  
fibrosis, or (3) a direct inhibition of \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .  
DRWD This invention provides for administering to an individual  
with a medical condition associated with \*\*\*TGF\*\*\* -.  
\*\*\*beta\*\*\* . excess a sufficient amount of \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .  
reduce -binding receptor fragment, such as s.beta.-RII, to  
\*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . activity in the individual.  
The \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . -binding receptor fragment is  
all or only a portion of a receptor which is capable of binding  
\*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . s.beta.-RII is made by  
synthesizing the extracellular domain of the \*\*\*TGF\*\*\* -.  
\*\*\*beta\*\*\* .

Type II receptor (.beta.-RII) and developing a fragment of  
this .beta.-RII domain as a high affinity, soluble binding  
protein (s.beta.-RII) for \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . This  
invention further provides for delivering s.beta.-RII to a site  
where \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . is in excess, such as in  
disease states characterized by fibroproliferation and  
\*\*\*immunosuppression\*\*\* such as is associated with  
infectious disease.  
DRWD s.beta.-RII fragments of the present invention  
may be used to treat viral infections in which there is an  
overproduction of \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . and  
\*\*\*immunosuppression\*\*\*. Examples of viruses with which \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .  
excess is associated include, but are not limited to,  
hepatitis C, lymphocytic choriomeningitis, human immunodeficiency virus  
(HIV), and human T. . .  
DRWD The s.beta.-RII of the present invention also may be used  
to increase the efficacy of vaccines. Because \*\*\*TGF\*\*\* -.  
\*\*\*beta\*\*\* . may cause \*\*\*immunosuppression\*\*\*, the  
administration of s.beta.-RII can counteract  
\*\*\*immunosuppression\*\*\* caused by \*\*\*TGF\*\*\* -.  
\*\*\*beta\*\*\* . and increase the vaccine recipient's immune response to  
the vaccine. s.beta.-RII should be particularly effective in  
administered \*\*\*immunosuppressed\*\*\* patients. s.beta.-RII may be  
before or concomitantly with the vaccine.  
DRWD The medical history may reveal facts which support a  
diagnosis of fibroproliferative disorder, collagen vascular disease,  
wound \*\*\*immunosuppression\*\*\*, or of potential for problematic  
healing, as in peritoneal adhesions following surgery, or  
restenosis of blood vessels after coronary angioplasty.  
Conditions which are identified as being associated with high levels  
of \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . and/or proliferation of  
fibrous tissue are considered to cause \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .  
excess.  
DETJ Injection of SCW produces an acute inflammatory response  
which is clinically detectable within hours and maximal in 3-5  
days. When anti- \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . is injected directly  
into a joint before ip administration of the SCW, inflammation at  
24 hours is significantly below that observed in joints with  
the irrelevant antibody. At the peak of the acute response,  
inflammation of anti- \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . joints  
remains far below that of joints with the irrelevant antibody.  
Even if joints are injected with anti- \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .  
when inflammation is well developed (day 13), anti- \*\*\*TGF\*\*\* -.  
\*\*\*beta\*\*\* . still has a significant \*\*\*anti\*\*\* -  
\*\*\*inflammatory\*\*\* effect, when compared to irrelevant  
antibody. Because s.beta.-RII also binds \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .  
s.beta.-RII has a similarly beneficial effect when given  
early or late in the inflammatory process.  
L14 ANSWER 8 OF 15 USPATFULL  
PI US 5688765 971118  
SUMM . . . to be unique and lacrimal gland-specific. It also  
appears that this hormone effect is not linked to a generalized,  
systemic \*\*\*anti\*\*\* - \*\*\*inflammatory\*\*\* function. Building on  
these new discoveries, the method of the invention involves a  
rejection of the classical therapeutic approach to. . . effects  
of systemic administration. Furthermore, since the  
androgen-induced suppression of lacrimal gland inflammation could be  
mediated through the induction of \*\*\*transforming\*\*\*  
\*\*\*growth\*\*\* \*\*\*factor\*\*\* -. \*\*\*beta\*\*\* . ( \*\*\*TGF\*\*\* -.  
\*\*\*beta\*\*\* .), a potent \*\*\*immunosuppressive\*\*\* compound, local  
application of \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . should also have the  
same effect.  
DETJ . . . be through the control of epithelial cell  
cytokine production. In support of this hypothesis, as has been  
described above, the \*\*\*anti\*\*\* - \*\*\*inflammatory\*\*\* of  
androgens in lacrimal tissue appears to be mediated not  
through lymphocytes, but rather through epithelial cells.  
Moreover, epithelial cells in other tissues are known to secrete  
numerous cytokines, e.g., \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . (137), and

also serve in exocrine sites as active cellular participants in the glandular inflammation in Sjogren's syndrome (138). In addition, as will be described below, androgens increase the mRNA and protein levels of the \*\*\*immunosuppressive\*\*\* cytokine, \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .1, in the lacrimal gland. This cytokine is thought to play a protective role in Sjogren's syndrome, and increased expression of \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .1 has been correlated with reduced inflammation in salivary glands of Sjogren's syndrome patients (129). In contrast, the absence of \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .1 leads to a pronounced lymphocytic infiltration into both lacrimal and salivary glands (139).

DETD . . . endocrine regulation of immune function in this tissue. These studies, which were conducted with high stringency, RT-PCR procedures, demonstrated that \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .1, \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .2, \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .3, IL-6, TNF-.alpha. and IL-1.alpha. mRNA may be detected consistently in lacrimal glands, as well as in isolated acinar epithelial . . . of male and female rats. As a corollary to these studies, whether lacrimal glands of autoimmune mice contain mRNAs for \*\*\*anti\*\*\* - \*\*\*inflammatory\*\*\* , as well as pro-inflammatory, cytokines was also examined. This research, which was performed with high stringency RT-PCR techniques, showed that mRNA for IL-1.alpha., IL-1.beta., IL-2 receptor, IL-6, \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . and TNF-.alpha. may be detected consistently in lacrimal glands of autoimmune female MRL/lpr mice.

DETD . . . in lacrimal tissues of normal rats and autoimmune mice. In addition, these results demonstrate that androgens stimulate the accumulation of \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .1 mRNA and protein in the lacrimal gland. \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .1, in turn, is known to exert profound \*\*\*immunosuppressive\*\*\* activity, including the inhibition of T and B cell proliferation, cytotoxic T cell generation, natural and lymphokine-activated killing, T cell . . . production, and is believed to down regulate inflammation in exocrine glands in Sjogren's syndrome (129,139,140). Consequently, the androgen-induced increase in \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .1 could act to suppress lymphocytic infiltration and to attenuate IL-1 and TNF-.alpha. production in the lacrimal gland. These hormonal effects. . .

L14 ANSWER 9 OF 15 USPATFULL  
PI US 5661179 970826  
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DETD . . . profound changes in tumor behavior were accompanied by alterations in the expression of genes implicated in growth control, angiogenesis, and \*\*\*immunosuppression\*\*\* (e.g., TGF.alpha., HbF, and \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .2).

DETD . . . athymic mice. Molecular analysis of brain and hormone-refractory prostate cancer cells revealed marked decline in the production and secretion of \*\*\*TGF\*\*\* .

\*\*\*beta\*\*\* . . . a protein implicated in growth control, angiogenesis, and \*\*\*immunosuppression\*\*\* . Treated prostatic cells exhibited decreased proteolytic activity mediated by urokinase-plasminogen activator, a molecular marker of disease progression in man.

DETD The malignant prostatic cell lines exhibit numerous abnormalities in gene expression, including increased production of autocrine tumor growth factor-.beta. ( \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .) and elevated activity of urokinase plasminogen activator (uPA). Members of the \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . family have been implicated in tumor growth control, angiogenesis, and \*\*\*immunosuppression\*\*\* . uPA, in contrast, is a serine protease involved in degradation of extracellular stroma and basal lamina structures, with the potential to facilitate tumor invasion and metastasis. It was of interest, therefore, to examine the effect of NaPA on \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . and uPA expression in the prostatic tumor cells. Northern blot analysis of PC3 after 72

h treatment revealed a decrease in \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .2 mRNA levels; the effect was specific for \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .2 as there was no change in the expression of \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .1. The decrease in \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .2 was accompanied by approximately a twofold increase in the levels of HLA-A3 mRNA, as previously observed in treated human leukemic. . .

L14 ANSWER 10 OF 15 USPATFULL  
PI US 5654333 970805  
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DETD . . . profound changes in tumor behavior were accompanied by alterations in the expression of genes implicated in growth control, angiogenesis, and \*\*\*immunosuppression\*\*\* (e.g., TGF.alpha., HbF, and \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .2).

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L14 ANSWER 11 OF 15 USPATFULL  
PI US 5635533 970603  
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DETD . . . profound changes in tumor behavior were accompanied by alterations in the expression of genes implicated in growth control, angiogenesis, and \*\*\*immunosuppression\*\*\* (e.g., TGF.alpha., HbF, and \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .2).

DETD . . . athymic mice. Molecular analysis of brain and hormone-refractory prostate cancer cells revealed marked decline in the production and secretion of \*\*\*TGF\*\*\* .

\*\*\*beta\*\*\* . . . a protein implicated in growth control, angiogenesis, and \*\*\*immunosuppression\*\*\* . Treated prostatic cells exhibited decreased proteolytic activity mediated by urokinase-plasminogen activator, a molecular marker of disease progression in man.

DETD The malignant prostatic cell lines exhibit numerous abnormalities in gene expression, including increased production of autocrine tumor growth factor-.beta. ( \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .) and elevated activity of urokinase plasminogen activator (uPA). Members of the \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . family have been implicated in tumor growth control, angiogenesis, and \*\*\*immunosuppression\*\*\* . uPA, in contrast, is a serine protease involved in degradation of extracellular stroma and basal lamina structures, with the potential to facilitate tumor invasion and metastasis. It was of interest, therefore, to examine the effect of NaPA on \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . and uPA expression in the prostatic tumor cells. Northern blot analysis of PC3 after 72

h treatment revealed a decrease in \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .2 mRNA levels; the effect was specific for \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .2 as there was no change in the expression of \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .1. The decrease in \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .2 was accompanied by approximately a twofold

increase in the levels of HLA-A3 mRNA, as previously observed in treated human leukemic. . .

L14 ANSWER 12 OF 15 USPATFULL  
PI US 563532 970603

DETD . . . profound changes in tumor behavior were accompanied by alterations in the expression of genes implicated in growth control, angiogenesis, and \*\*\*immunosuppression\*\*\* (e.g., TGF.alpha., HbF, and \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .2).

DETD . . . athymic mice. Molecular analysis of brain and hormone-refractory prostate cancer cells revealed marked decline in the production and secretion of \*\*\*TGF\*\*\* .

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L14 ANSWER 13 OF 15 USPATFULL  
PI US 5605930 970225

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of protein \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .2, coding for a 12.5-kD implicated in glioma autocrine growth, angiogenesis, and tumor-induced \*\*\*immunosuppression\*\*\* . Synergy between NaPA and LOV could be due to the ability of each to block the MVA pathway at distinct. . .

DETD The \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . autocrine growth regulatory pathways are of particular interest in primary central nervous system tumors. This pluripotential growth regulator is produced by both primary malignant astrocytoma tissue (Clark WC, Bressler J: \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .-like activity in tumors of the central nervous system. J Neurosurg 68: 920-924, 1988; Samuels V, Barrett JM, Brochman Set al.: . . . Pathol 134: 895-902, 1989) and by cell lines derived from such tumors (Jennings MT, Macina RJ, Carver R et al.: \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .1 and \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .2 are potential growth regulators for low grade and malignant gliomas in vitro with evidence in support of an autocrine hypothesis. Int. J. Cancer 49: 129-139, 1991).

The \*\*\*immunosuppressive\*\*\* effects of malignant astrocytoma cells on cocultured lymphocytes in vitro has been convincingly linked to \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . production and can be partially neutralized by antibodies against \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .

\*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . has been shown to be a growth regulator for gliomas in vitro (Jennings MT, Macina RJ, Carver R et al.: \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .1 and \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .2 are potential growth regulators for low grade and malignant gliomas in vitro with evidence in support of an autocrine hypothesis. Int. J. Cancer 49: 129-139, 1991).

The role of the \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . pathway in growth regulation of medulloblastoma is less well established than for malignant astrocytomas. The antiproliferative effect of ATRA on Daoy medulloblastoma cells is associated with increased secretion of \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* .2 and with induction of \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . receptor expression. Because the \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . family of growth factors plays such an important role in the biology of malignant astrocytomas, the initial focus has been. . .

L14 ANSWER 14 OF 15 USPATFULL  
PI US 5268455 931207

AB A polypeptide is provided that excludes (a) a full-length mature \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . molecule or precursor \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . molecule or deletion variants of mature or precursor \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . molecules in which from about 1 to 10 amino acid residues have been deleted, (b) a polypeptide of the sequence: . . . the sequence: Arg-Asn-Leu-Glu-Glu-Asn-Cys-Val-Arg-Pro-Leu-Tyr-Ile-Asp-Phe-Arg-Gln-Asp-Leu, the polypeptide comprising an amino acid sequence that is based on conserved sequences in the family of \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . molecules. Such polypeptides are particularly useful therapeutically as \*\*\*immunosuppressive\*\*\* agents when coupled to carrier proteins or crosslinked to form polymers.

SUMM \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . has been shown to have numerous regulatory actions on a wide variety of both normal and neoplastic cells. Recent studies indicate an important role for \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . in cells of the immune system (J. Kehrl et al., J. Exp. Med., 163:1037 [1986]; H-J. Ristow, Proc. Natl. Acad. Sci. U.S.A., 83:2438 [1986]; C. Shipley et al. Cancer Res., 46:2068 [1986]).

Moreover, \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . has been described as a suppressor of cytokine (e.g., IFN-.gamma., TNF-.alpha.) production, indicating its use as an \*\*\*immunosuppressant\*\*\* for treating inflammatory disorders (Espevik et al., J. Exp. Med., 166:571-576 [1987]; European Pat. Pub. No. 269,408 published Jun. 1, 1987, issued Feb. 21, 1989), and as a promoter of cachexia (Beutler and Cerami, New Eng. J. Med., 316:379 [1987]). Further, \*\*\*TGF\*\*\* -. \*\*\*beta\*\*\* . induces collagen secretion in human fibroblast cultures (Roberts et al., Proc. Nat. Acad. Sci. USA 83:4167-4171

(1986); Chua et al., . . . . .  
 SUMM . . . . . a novel class of immune modulators that are  
 useful to develop diagnostic assays for the presence in patient  
 fluids of \*\*\*immunosuppressive\*\*\* proteins such as \*\*\*TGF\*\*\* .  
 \*\*\*beta\*\*\* . . . or antibodies to such proteins.  
 SUMM In a still further embodiment, the invention comprises a  
 method for producing antibodies that neutralize  
 \*\*\*immunosuppressive\*\*\* proteins comprising immunizing an animal with the  
 polypeptide identified above that has the sequence containing the X  
 moiety and isolating antibodies generated by the polypeptide that  
 neutralize at least one \*\*\*immunosuppressive\*\*\* protein,  
 preferably \*\*\*TGF\*\*\* . . . \*\*\*beta\*\*\* .  
 DETD As used herein, the term " \*\*\*immunosuppressive\*\*\*  
 protein" as used in the context that it is neutralized by antibodies  
 generated by the polypeptide herein generally refers to the protein  
 to which the polypeptide corresponds that exhibits  
 \*\*\*immunosuppressive\*\*\* activity. For example, a  
 polypeptide representing an internal, active sequence within the  
 full-length \*\*\*TGF\*\*\* . . . \*\*\*beta\*\*\* . sequence would have  
 \*\*\*TGF\*\*\* . . . \*\*\*beta\*\*\* . as its \*\*\*immunosuppressive\*\*\* protein  
 to be neutralized.  
 DETD "Biologically active" polypeptides herein are defined as  
 those having the ability to cross-react with antisera raised  
 against native \*\*\*TGF\*\*\* . . . \*\*\*beta\*\*\* . (where native  
 \*\*\*TGF\*\*\* . . . \*\*\*beta\*\*\* . is that which is obtained from platelets  
 or other natural sources). Immunological crossreactivity is a  
 measure of a single active epitope and does not necessarily  
 encompass an active domain involved in \*\*\*immunosuppressive\*\*\*  
 activity.  
 DETD . . . peptide neutralizes monoclonal and polyclonal  
 antibodies raised against a corresponding native protein known to  
 have biological/therapeutic activity, e.g., mature human  
 \*\*\*TGF\*\*\* . . . \*\*\*beta\*\*\* . . Other indications, specific for the  
 \*\*\*TGF\*\*\* . . . \*\*\*beta\*\*\* . polypeptide herein, include whether the  
 peptide stimulates release of PGE2 by IL-1 treated human  
 fibroblasts, interferes with the binding of full-length \*\*\*TGF\*\*\* . .  
 \*\*\*beta\*\*\* . to its receptors, or acts as an  
 \*\*\*immunosuppressive\*\*\* agent either in vitro or in vivo  
 in an animal model. The first step, then, involves determining  
 that the monomeric . . .  
 DETD Since the polypeptides herein are, in general, related to  
 \*\*\*immunosuppressive\*\*\* proteins, they are also useful  
 as immunogens to elicit antibodies capable of blocking the  
 \*\*\*immunosuppressive\*\*\* activity associated with or  
 caused by such \*\*\*immunosuppressive\*\*\* proteins, e.g.,  
 \*\*\*TGF\*\*\* . . . \*\*\*beta\*\*\* . . Examples of such activity include  
 neoplastic or viral disorders. For making immunogenic peptides capable  
 of eliciting antibodies to the \*\*\*immunosuppressive\*\*\*  
 proteins, the polypeptides are typically not  
 \*\*\*immunosuppressive\*\*\*, either because they are in monomeric form or because they  
 are modified to be so. This modification can be performed by  
 substituting one or more of the amino acids within the  
 polypeptide polymer sequence to obtain non- \*\*\*immunosuppressive\*\*\*  
 immunogenic forms of the polypeptides. The proper amino  
 acids to be modified can be tested simply by making the  
 substitution. . . .  
 L14 ANSWER 15 OF 15 USP24FULL  
 PI US 5061786 911029  
 <--  
 AB A polypeptide is provided that excludes (a) a full-length  
 mature \*\*\*TGF\*\*\* . . . \*\*\*beta\*\*\* . molecule or precursor  
 \*\*\*TGF\*\*\* . . . \*\*\*beta\*\*\* . molecule or deletion variants of mature or  
 precursor \*\*\*TGF\*\*\* . . . \*\*\*beta\*\*\* . molecules in which from about  
 1 to 10 amino acid residues have been deleted, (b) a polypeptide  
 of the sequence: . . . sequence: Arg  
 Asn-Leu-Glu-Glu-Asn-Cys-Cys-Val-  
 Arg-Pro-Leu-Tyr-Ile-Asp-Phe-Arg-Gln-Asp-Leu, said  
 polypeptide comprising an amino acid sequence that is based on  
 conserved sequences in the family of \*\*\*TGF\*\*\* . . . \*\*\*beta\*\*\* .  
 molecules. Such polypeptides are particularly useful  
 therapeutically as \*\*\*immunosuppressive\*\*\* agents when  
 coupled to carrier proteins or crosslinked to form polymers.  
 SUMM \*\*\*TGF\*\*\* . . . \*\*\*beta\*\*\* . has been shown to have

numerous regulatory actions on a wide variety of both normal and  
 neoplastic cells. Recent studies indicate an important role for  
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 et al., J. Exp. Med. 163:1037 [1986]; H-J Ristow, Proc. Natl.  
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 SUMM . . . . . a novel class of immune modulators that are  
 useful to develop diagnostic assays for the presence in patient  
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 \*\*\*beta\*\*\* . . or antibodies to such proteins.  
 SUMM In a still further embodiment, the invention comprises a  
 method for producing antibodies that neutralize  
 \*\*\*immunosuppressive\*\*\* proteins comprising immunizing an animal with the  
 polypeptide identified above that has the sequence containing the X  
 moiety and isolating antibodies generated by the polypeptide that  
 neutralize at least one \*\*\*immunosuppressive\*\*\* protein,  
 preferably \*\*\*TGF\*\*\* . . . \*\*\*beta\*\*\* .  
 DETD As used herein, the term " \*\*\*immunosuppressive\*\*\*  
 protein" as used in the context that it is neutralized by antibodies  
 generated by the polypeptide herein generally refers to the protein  
 to which the polypeptide corresponds that exhibits  
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 polypeptide representing an internal, active sequence within the  
 full-length \*\*\*TGF\*\*\* . . . \*\*\*beta\*\*\* . sequence would have  
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 to be neutralized.  
 DETD "Biologically active" polypeptides herein are defined as  
 those having the ability to cross-react with antisera raised  
 against native \*\*\*TGF\*\*\* . . . \*\*\*beta\*\*\* . (where native  
 \*\*\*TGF\*\*\* . . . \*\*\*beta\*\*\* . is that which is obtained from platelets  
 or other natural sources). Immunological cross-reactivity is  
 a measure of a single active epitope and does not  
 necessarily encompass an active domain involved in  
 \*\*\*immunosuppressive\*\*\* activity.  
 DETD . . . peptide neutralizes monoclonal and polyclonal  
 antibodies raised against a corresponding native protein known to  
 have biological/therapeutic activity, e.g., mature human  
 \*\*\*TGF\*\*\* . . . \*\*\*beta\*\*\* . . Other indications, specific for the  
 \*\*\*TGF\*\*\* . . . \*\*\*beta\*\*\* . polypeptide herein, include whether the  
 peptide stimulates release of PGE2 by IL-1 treated human  
 fibroblasts, interferes with the binding of full-length \*\*\*TGF\*\*\* . .  
 \*\*\*beta\*\*\* . to its receptors, or acts as an  
 \*\*\*immunosuppressive\*\*\* agent either in vitro or in vivo  
 in an animal model. The first step, then, involves determining  
 that the monomeric . . .  
 DETD Since the polypeptides herein are, in general, related to  
 \*\*\*immunosuppressive\*\*\* proteins, they are also useful  
 as immunogens to elicit antibodies capable of blocking the  
 \*\*\*immunosuppressive\*\*\* activity associated with or  
 caused by such \*\*\*immunosuppressive\*\*\* proteins, e.g.,  
 \*\*\*TGF\*\*\* . . . \*\*\*beta\*\*\* . . Examples of such activity include  
 neoplastic or viral disorders. For making immunogenic peptides capable  
 of eliciting antibodies to the \*\*\*immunosuppressive\*\*\*  
 proteins, the polypeptides are typically not  
 \*\*\*immunosuppressive\*\*\*, either because they are in monomeric form or because they  
 are modified to be so. This modification can be performed by  
 substituting one or more of the amino acids within the  
 polypeptide polymer sequence to obtain non- \*\*\*immunosuppressive\*\*\*  
 immunogenic forms of the polypeptides. The proper amino

acids to  
be modified can be tested simply by making the  
substitution.

=> d his

(FILE 'HOME' ENTERED AT 17:13:09 ON 15 AUG 1998)

FILE 'USPATFULL' ENTERED AT 17:14:49 ON 15 AUG 1998

L1 380 S CHRONIC RENAL FAILURE  
L2 18112 S ANTIINFLAM? OR (ANTI INFLAMMAT?) OR  
L3 IMMUNOSUPPRESS?  
L4 845 S (BONE(W)MORPHOGEN?) OR BMP? OR  
L5 (OSTEOGENIC(W)(PROTEIN?)  
L6 1271 S TGF $\beta$ TA## OR (TGF(W) $\beta$ TA##) OR  
L7 ((TRANSFORMING(W)GROWTH(  
L8 26 S L2(P)L4  
L9 109 S L2(P)L5  
L10 0 S L1 AND L6  
L11 0 S L1 AND L7  
L12 E GLOMERULO?  
L13 1114 S E8-E29  
L14 0 S L10 AND L6  
L15 15 S L10 AND L7  
L16 SELECT L12 1-15 PN  
L17 15 S E1-E15  
L18 15 S L13 AND L7

=> s l13 and l10

L15 15 L13 AND L10

=> d kwic 1-15

L15 ANSWER 1 OF 15 USPATFULL  
PI US 5759836 980602

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SUMM . . . NO, and also decreasing the levels of PGEs. In  
the MRL-lpr/lpr mutant mouse, NOS inhibitors prevent anti-DNA  
immune complex and \*\*\*glomerulonephritis\*\*\*, and reduce the  
intensity of inflammatory arthritis (Weinberg et al., J. Exp. Med.  
179:651-660, 1994).

L15 ANSWER 2 OF 15 USPATFULL  
PI US 5747532 980505

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DETD . . . disease), diabetes, arthritis, asthma,  
Alzheimer's disease, Parkinson's disease, multiple sclerosis,  
cirrhosis, allograft rejection, encephalomyelitis, meningitis,  
pancreatitis, peritonitis, vasculitis, lymphocytic choriomeningitis,  
\*\*\*glomerulonephritis\*\*\*, uveitis, ileitis, liver  
inflammation, renal inflammation, hemorrhagic shock, anaphylactic shock,  
burn, infection (including bacterial, viral, fungal and  
parasitic infections), hemodialysis, . . .

L15 ANSWER 3 OF 15 USPATFULL  
PI US 5712307 980127

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DETD . . . including (1) non-malignant disorders associated  
with abnormal differentiation programs, autoimmunity and  
inflammatory processes, e.g., rheumatoid arthritis, Castleman's  
disease, mesangial proliferation, \*\*\*glomerulonephritis\*\*\*,  
uveitis, sepsis, autoimmune diseases such as lupus, inflammatory  
bowel, type I diabetes, vasculitis, and several skin disorders of  
cell differentiation. . .

L15 ANSWER 4 OF 15 USPATFULL  
PI US 5710178 980120

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DETD . . . including (1) non-malignant disorders associated  
with abnormal differentiation programs, autoimmunity and  
inflammatory processes, e.g., rheumatoid arthritis, Castleman's  
disease, mesangial proliferation, \*\*\*glomerulonephritis\*\*\*,  
uveitis, sepsis, autoimmune diseases such as lupus, inflammatory  
bowel, type I diabetes, vasculitis, and several skin disorders of  
cell differentiation. . .

L15 ANSWER 5 OF 15 USPATFULL  
PI US 5708025 980113

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DETD . . . including (1) non-malignant disorders associated  
with abnormal differentiation programs, autoimmunity and  
inflammatory processes, e.g., rheumatoid arthritis, Castleman's  
disease, mesangial proliferation, \*\*\*glomerulonephritis\*\*\*,  
uveitis, sepsis, autoimmune diseases such as lupus, inflammatory  
bowel, type I diabetes, vasculitis, and several skin disorders of  
cell differentiation. . .

L15 ANSWER 6 OF 15 USPATFULL  
PI US 5693610 971202

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SUMM . . . are involved in various diseases and symptoms of  
immunosuppression in a cancer patient or the like, fibroid  
lung, hepatic fibrosis, \*\*\*glomerulonephritis\*\*\*,  
scleroderma, or the like. Further, PDGF acts on smooth muscle cells,  
fibroblasts, nerve gliocytes or the like to promote their. . .  
CLM What is claimed is:  
. . . and which contains about 18 to 38% by weight of  
protein, said  
disease or condition being fibroid lung, hepatic fibrosis,  
\*\*\*glomerulonephritis\*\*\*, or scleroderma.

L15 ANSWER 7 OF 15 USPATFULL  
PI US 5693607 971202

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SUMM . . . degrade these proteins. Thus, TGF- $\beta$  can  
cause fibrous tissue to accumulate. For example, in diabetic  
nephropathy and human mesangial proliferative  
\*\*\*glomerulonephritis\*\*\*, both fibroproliferative diseases, a prominent and  
important pathological feature is the accumulation of mesangial  
matrix (Mauer et al. (1984) J. . .  
SUMM Border et al. (1990) Nature 346:371-74, found that  
antiserum against TGF- $\beta$  suppressed experimentally induced  
\*\*\*glomerulonephritis\*\*\*, which was characterized by  
mesangial proliferation. Border et al. reported that the antibodies  
to TGF- $\beta$  which were raised in rabbits. . .  
SUMM Another way of suppressing TGF- $\beta$  in experimental  
\*\*\*glomerulonephritis\*\*\* in rats, which is associated  
with TGF- $\beta$  excess, was a low-protein diet. Both the  
excreted nitrogen and the expressed TGF- $\beta$ 1.  
SUMM . . . hepatic, intraocular and pulmonary fibrosis. In a  
further embodiment, the TGF- $\beta$  receptor fragment is  
administered to patients with diabetic nephropathy,  
\*\*\*glomerulonephritis\*\*\*, proliferative vitreoretinopathy, rheumatoid arthritis,  
liver cirrhosis, and biliary fibrosis.  
DRWD . . . administration of s. $\beta$ -RII fragments of the  
present invention may be used in fibroproliferative disorders. As  
mentioned above, animal models of  
\*\*\*glomerulonephritis\*\*\* have shown good results with anti-TGF- $\beta$  antibodies  
blocking excess TGF- $\beta$ . These antibodies will be difficult to  
deliver because they have. . . Thus, it would be preferable to  
close administer a lower molecular weight, native protein or  
analog, such as s. $\beta$ -RII, in \*\*\*glomerulonephritis\*\*\*.  
but are Kidney diseases associated with TGF- $\beta$  excess include,  
\*\*\*glomerulonephritis\*\*\* not limited to, mesangial proliferative  
\*\*\*glomerulonephritis\*\*\*, crescentic \*\*\*glomerulonephritis\*\*\*, diabetic  
nephropathy, renal interstitial fibrosis, renal fibrosis in transplant  
patients receiving cyclosporin, and HIV-associated nephropathy.  
These conditions are associated with. . .  
DRWD Systemic administration is the preferred mode of  
administration in \*\*\*glomerulonephritis\*\*\*, liver cirrhosis,  
immunosuppressive conditions (such as viral infections, AIDS and  
trypanosomal infections), and in widespread skin diseases (such as  
progressive.  
DETD The effect of s. $\beta$ -RII is compared with that of  
anti-TGF antibody in a \*\*\*glomerulonephritis\*\*\* model.  
Experimental \*\*\*glomerulonephritis\*\*\* can be induced in rats with a  
single injection of antithymocyte serum because the glomerular  
mesangial cells express a thy-1.1 epitope on their surfaces. The  
experimental lesion is acute mesangial proliferative  
\*\*\*glomerulonephritis\*\*\* and is characterized by  
expansion of the mesangial matrix and hypercellularity. The injured  
cells also express more TGF- $\beta$ 1 mRNA and.  
DETD First, \*\*\*glomerulonephritis\*\*\* is induced in rats by  
an intravenous injection of antithymocyte serum. Next, for  
six days, three groups of rats are. . .  
DETD . . . the kidneys, which are stained with periodic  
acid-Schiff solution to emphasize the pathological changes. The  
negative control kidneys have full-blown \*\*\*glomerulonephritis\*\*\*  
with reddish-pink amorphous fibrous material filling most of  
the glomerulus. The positive control kidneys have a staining  
pattern which is. . .  
CLM What is claimed is:  
5. The method of claim 3 wherein said fibroproliferative  
disorder is selected from the group consisting of diabetic



nephropathy,  
\*\*\*glomerulonephritis\*\*\* , proliferative  
vitreoretinopathy, liver  
cirrhosis, biliary fibrosis, and myelofibrosis.

L15 ANSWER 8 OF 15 USPATFULL  
PI US 5688765 971118

DETD . . . S., Goodman, J. R., and Siiteri, P. K., Effect of  
castration and sex-hormone treatment on survival,  
anti-nucleic  
acid antibodies, and \*\*\*glomerulonephritis\*\*\* in  
NZB/NZW FI  
mice. J. Exp. Med. 147:1568-1583 (1978).

L15 ANSWER 9 OF 15 USPATFULL  
PI US 5661179 970826

DETD . . . including (1) non-malignant disorders associated  
with  
abnormal differentiation programs, autoimmunity and  
inflammatory  
processes, e.g., rheumatoid arthritis, Castleman's  
disease,  
mesangial proliferation, \*\*\*glomerulonephritis\*\*\* ,  
uveitis,  
sepsis, autoimmune diseases such as lupus, inflammatory  
bowel,  
type I diabetes, vasculitis, and several skin disorders of  
cell  
differentiation. . .

L15 ANSWER 10 OF 15 USPATFULL  
PI US 5654333 970805

DETD . . . including (1) non-malignant disorders associated  
with  
abnormal differentiation programs, autoimmunity and  
inflammatory  
processes, e.g., rheumatoid arthritis, Castleman's  
disease,  
mesangial proliferation, \*\*\*glomerulonephritis\*\*\* ,  
uveitis,  
sepsis, autoimmune diseases such as lupus, inflammatory  
bowel,  
type I diabetes, vasculitis, and several skin disorders of  
cell  
differentiation. . .

L15 ANSWER 11 OF 15 USPATFULL  
PI US 5635533 970603

DETD . . . including (1) non-malignant disorders associated  
with  
abnormal differentiation programs, autoimmunity and  
inflammatory  
processes, e.g., rheumatoid arthritis, Castleman's  
disease,  
mesangial proliferation, \*\*\*glomerulonephritis\*\*\* ,  
uveitis,  
sepsis, autoimmune diseases such as lupus, inflammatory  
bowel,  
type I diabetes, vasculitis, and several skin disorders of  
cell  
differentiation. . .

L15 ANSWER 12 OF 15 USPATFULL  
PI US 5635532 970603

DETD . . . including (1) non-malignant disorders associated  
with  
abnormal differentiation programs, autoimmunity and  
inflammatory  
processes, e.g., rheumatoid arthritis, Castleman's  
disease,  
mesangial proliferation, \*\*\*glomerulonephritis\*\*\* ,  
uveitis,  
sepsis, autoimmune diseases such as lupus, inflammatory  
bowel,  
type I diabetes, vasculitis, and several skin disorders of  
cell  
differentiation. . .

L15 ANSWER 13 OF 15 USPATFULL  
PI US 5605930 970225

SUMM  
of the . . . of inhibiting may be used in a subject having any  
following pathologies: rheumatoid arthritis, Castleman's  
disease,  
mesangial proliferation, \*\*\*glomerulonephritis\*\*\* ,  
uveitis,  
sepsis, autoimmunity inflammatory bowel, type I diabetes,  
vasculitis and a cell differentiation associated skin  
disorder.

DETD . . . of inhibiting may be used in a subject having any  
of the  
following pathologies: rheumatoid arthritis, Castleman's  
disease,  
mesangial proliferation, \*\*\*glomerulonephritis\*\*\* ,  
uveitis,  
sepsis, autoimmunity inflammatory bowel, type I diabetes,  
vasculitis and a cell differentiation associated skin  
disorder.

DETD . . . including (1) non-malignant disorders associated  
with  
abnormal differentiation programs, autoimmunity and  
inflammatory  
processes, e.g., rheumatoid arthritis, Castleman's  
disease,  
mesangial proliferation, \*\*\*glomerulonephritis\*\*\* ,  
uveitis,  
sepsis, autoimmune diseases such as lupus, inflammatory  
bowel,  
type I diabetes, vasculitis, and several skin disorders of  
cell  
differentiation. . .

L15 ANSWER 14 OF 15 USPATFULL  
PI US 5268455 931207

DETD . . . lupus erythematosus, rheumatoid arthritis,  
scleroderma,  
dermatomyositis, polymyositis, unclassified connective  
diseases,  
autoimmune hemolytic anemia, idiopathic thrombocytopenic  
purpura,  
autoimmune thyroiditis, polyarteritis nodosum,  
\*\*\*glomerulonephritis\*\*\* , uveitis, etc.

L15 ANSWER 15 OF 15 USPATFULL  
PI US 5061786 911029

DETD . . . lupus erythematosus, rheumatoid arthritis,  
scleroderma,  
dermatomyositis, polymyositis, unclassified connective  
diseases,  
autoimmune hemolytic anemia, idiopathic thrombocytopenic  
purpura,  
autoimmune thyroiditis, polyarteritis nodosum,  
\*\*\*glomerulonephritis\*\*\* , uveitis, etc.

=> file medline

COST IN U.S. DOLLARS  
TOTAL

SINCE FILE

SESSION  
FULL ESTIMATED COST  
18.30

ENTRY

17.85

FILE 'MEDLINE' ENTERED AT 17:24:48 ON 15 AUG 1998

FILE LAST UPDATED: 11 AUG 1998 (19980811/UP). FILE COVERS 1966  
TO DATE.

THE MEDLINE FILE WAS RELOADED FEBRUARY 15, 1998, TO REFLECT THE  
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MESH (MEDICAL SUBJECT HEADING) CHANGES. ENTER HELP RLOAD FOR  
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THIS FILE CONTAINS CAS REGISTRY NUMBERS FOR EASY AND ACCURATE  
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=> d his

(FILE 'HOME' ENTERED AT 17:13:09 ON 15 AUG 1998)

FILE 'USPATFULL' ENTERED AT 17:14:49 ON 15 AUG 1998

L1 380 S CHRONIC RENAL FAILURE  
L2 18112 S ANTIINFLAM? OR (ANTI INFLAMMAT?) OR  
IMMUNOSUPPRESS?  
L3 78 S L1 AND L2  
L4 845 S (BONE(W)MORPHOGEN?) OR BMP? OR  
(OSTEOGENIC(W)(PROTEIN?  
L5 1271 S TGFbeta## OR (TGF(W)BETA##) OR  
(TRANSFORMING(W)GROWTH(  
L6 26 S L2(P)L4  
L7 109 S L2(P)L5  
L8 0 S L1 AND L6  
L9 0 S L1 AND L7  
E GLOMERULO?  
L10 1114 S E8-E29  
L11 0 S L10 AND L6  
L12 15 S L10 AND L7  
SELECT L12 1-15 PN  
L13 15 S E1-E15  
L14 15 S L13 AND L7  
L15 15 S L13 AND L10

FILE 'MEDLINE' ENTERED AT 17:24:48 ON 15 AUG 1998

=> s l1 and l2

354059 CHRONIC  
210701 RENAL  
211454 FAILURE  
10652 CHRONIC RENAL FAILURE  
(CHRONIC(W)RENAL(W)FAILURE)  
5575 ANTIINFLAM?  
263229 ANTI  
154247 INFLAMMAT?  
37381 ANTI INFLAMMAT?  
(ANTI(W)INFLAMMAT?)  
60980 IMMUNOSUPPRESS?  
L16 232 L1 AND L2

=> s l16 and l4

277336 BONE  
19348 MORPHOGEN?  
1506 BONE(W)MORPHOGEN?  
1262 BMP?  
3474 OSTEOGENIC  
1019390 PROTEIN?  
66695 POLYPEPTIDE?  
113 OSTEOGENIC(W)(PROTEIN? OR POLYPEPTIDE?)  
L17 0 L16 AND L4

=> s l16 and l5

293 TGFbeta##  
11091 TGF  
327114 BETA##  
8923 TGF(W)BETA##  
24093 TRANSFORMING  
467672 GROWTH  
1400980 FACTOR#  
327114 BETA##  
11190 (TRANSFORMING(W)GROWTH(W)FACTOR#)(1A)BETA##  
L18 1 L16 AND L5

=> d au ti so

L18 ANSWER 1 OF 1 MEDLINE  
AU Shimizu T; Fukagawa M; Kuroda T; Hata S; Iwasaki Y; Nemoto  
M; Shirai



K; Yamauchi S; Margolin S B; Shimizu F; Kurokawa K  
TI Pirfenidone prevents collagen accumulation in the remnant  
kidney in  
rats with partial nephrectomy.  
SO KIDNEY INTERNATIONAL. SUPPLEMENT, (1997 Dec) 63 S239-43.  
Journal code: KVC. ISSN: 0098-6577.

=> d ab

L18 ANSWER 1 OF 1 MEDLINE  
AB Pirfenidone (PFD) is a new compound that prevents and even  
reverses  
the extracellular matrix accumulation in several organs as  
shown by  
experimental and clinical studies. In the present study, we  
examined  
the effect of PFD (500 mg/kg daily in the food) on the  
progression  
of \*\*\*chronic\*\*\* \*\*\*renal\*\*\* \*\*\*failure\*\*\* (CRF)  
in the  
5/6 nephrectomy rat model. Proteinuria progressively  
increased in  
rats with renal ablation (C) at 12 weeks. Urinary protein  
excretion  
in PFD-treated rats (P) was numerically lower than C, but  
the  
difference did not reach statistical significance. In  
contrast, in  
the chronic phase, PFD improved renal function and reduced  
collagen  
accumulation detected by hydroxyproline content (OH-Pro) in  
the  
cortex of the remnant kidney. Although creatinine clearance  
decreased with time in C, the values in P were significantly  
better  
at 10 and 12 weeks. The OH-Pro in C at 12 weeks was  
significantly  
higher than that of no-ablation, sham-operated rats, whereas  
OH-Pro  
in CRF was lower in (P). Expression of mRNA for type IV and  
I  
collagen in the cortex also increased in C, but it was  
inhibited in  
(P). To study the role that \*\*\*TGF\*\*\* - \*\*\*beta\*\*\*  
plays in  
the regulatory process following CRF, we examined the  
expression of  
\*\*\*TGF\*\*\* - \*\*\*beta\*\*\* mRNA in this model. Levels of  
cortical  
\*\*\*TGF\*\*\* - \*\*\*beta\*\*\* mRNA in C were significantly  
elevated at  
12 weeks. The increase was suppressed by PFD. These results  
demonstrate that PFD attenuates the development of CRF by  
preventing  
collagen accumulation in this model, and suggest that PFD  
can be  
clinically useful for treating CRF.

=> d his

(FILE 'HOME' ENTERED AT 17:13:09 ON 15 AUG 1998)

FILE 'USPATFULL' ENTERED AT 17:14:49 ON 15 AUG 1998

L1 380 S CHRONIC RENAL FAILURE  
L2 18112 S ANTIINFLAMM? OR (ANTI INFLAMMAT?) OR  
IMMUNOSUPPRESS?  
L3 78 S L1 AND L2  
L4 845 S (BONE(W)MORPHOGEN?) OR BMP? OR  
(OSTEOGENIC(W) (PROTEIN?)  
L5 1271 S TGF(BETA## OR (TGF(W)BETA##) OR  
(TRANSFORMING(W)GROWTH(  
L6 26 S L2(P)L4  
L7 109 S L2(P)L5  
L8 0 S L1 AND L6  
L9 0 S L1 AND L7  
L10 1114 S E8-E29  
L11 0 S L10 AND L6  
L12 15 S L10 AND L7  
L13 SELECT L12 1-15 PN  
L14 15 S E1-E15  
L15 15 S L13 AND L7  
L16 15 S L13 AND L10

FILE 'MEDLINE' ENTERED AT 17:24:48 ON 15 AUG 1998

L16 232 S L1 AND L2  
L17 0 S L16 AND L4  
L18 1 S L16 AND L5

=> file caplus

COST IN U.S. DOLLARS	SINCE FILE
TOTAL	ENTRY
SESSION	
FULL ESTIMATED COST	2.50
20.80	

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FILE COVERS 1967 - 15 Aug 1998 VOL 129 ISS 8  
FILE LAST UPDATED: 15 Aug 1998 (980815/ED)

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=> s l16; s l19 and l4; s l19 and l5

91316 CHRONIC  
81273 RENAL  
88438 FAILURE  
2950 CHRONIC RENAL FAILURE  
(CHRONIC(W)RENAL(W)FAILURE)

28488 ANTIINFLAMM?  
174425 ANTI  
83266 INFLAMMAT?  
14854 ANTI INFLAMMAT?  
(ANTI(W)INFLAMMAT?)

L19 25193 IMMUNOSUPPRESS?  
23 L1 AND L2

84342 BONE  
12643 MORPHOGEN?  
1542 BONE(W)MORPHOGEN?  
1587 BMP?  
1457 OSTEOGENIC  
1119373 PROTEIN?  
91655 POLYPEPTIDE?  
146 OSTEOGENIC(W) (PROTEIN? OR POLYPEPTIDE?)  
0 L19 AND L4

L20 846 TGF(BETA##  
11277 TGF  
762216 BETA##  
9132 TGF(W)BETA##  
29410 TRANSFORMING  
692464 GROWTH  
758204 FACTOR#  
762216 BETA##  
11087 (TRANSFORMING(W)GROWTH(W)FACTOR#) (1A)BETA##  
0 L19 AND L5

L21 0 L19 AND L5

=> d au ti so 1-23 l19

L19 ANSWER 1 OF 23 CAPLUS COPYRIGHT 1998 ACS  
AU Tamimi, N. A.; Stevens, P. E.; O'Donnell, P. L.; Strange, P.  
G.;

TI Muchaneta-Kubara, E. C.; El Nahas, A. M.  
Expression of cytoskeletal proteins differentiates between  
progressors and non-progressors in treated idiopathic  
membranous  
nephropathy  
SO Exp. Nephrol. (1998), 6(3), 217-225  
CODEN: EXNEEG; ISSN: 1018-7782

L19 ANSWER 2 OF 23 CAPLUS COPYRIGHT 1998 ACS  
AU Delzell, Elizabeth; Shapiro, Samuel  
TI A review of epidemiologic studies of nonnarcotic analgesics  
and  
chronic renal disease  
SO Medicine (Baltimore) (1998), 77(2), 102-121  
CODEN: MEDIAB; ISSN: 0025-7974

L19 ANSWER 3 OF 23 CAPLUS COPYRIGHT 1998 ACS  
AU Fillastre, Jean-Paul  
TI Ochratoxin-induced animal and human nephrotoxicity  
SO Bull. Acad. Natl. Med. (Paris) (1997), 181(7), 1447-1463  
CODEN: BANMAC; ISSN: 0001-4079

L19 ANSWER 4 OF 23 CAPLUS COPYRIGHT 1998 ACS  
AU Takeuchi, H.; Hirano, T.; Oka, K.; Mizumoto, K.; Akashi, T.;  
Sakurai, E.; Degawa, T.; Uchiyama, M.; Kozaki, K.; Matsuno,  
N.;

TI Nagao, T.; Kozaki, M.  
Lymphocyte sensitivity to cyclosporine and tacrolimus in  
\*\*\*chronic\*\*\* \*\*\*renal\*\*\* \*\*\*failure\*\*\* patients  
and  
clinical significance in renal transplantation  
SO Transplant. Proc. (1998), 30(1), 36-39  
CODEN: TRPPAB; ISSN: 0041-1345

L19 ANSWER 5 OF 23 CAPLUS COPYRIGHT 1998 ACS  
AU Smith, O. P.; White, B.; Vaughan, D.; Rafferty, M.; Claffey,  
L.;

TI Lyons, B.; Casey, W.  
Use of protein-C concentrate, heparin, and hemodiafiltration  
in  
Meningococcus-induced purpura fulminans  
SO Lancet (1997), 350(9091), 1590-1593  
CODEN: LANCAO; ISSN: 0140-6736

L19 ANSWER 6 OF 23 CAPLUS COPYRIGHT 1998 ACS  
AU Morrone, Luigi F.; Paolo, Salvatore Di; Logoluso, Francesco;  
Schna,  
Antonio; Stallone, Giovanni; Giorgino, Francesco; Schna, F.  
Paolo  
TI Interference of angiotensin-converting enzyme inhibitors on  
erythropoiesis in kidney transplant recipients: role of  
growth  
factors and cytokines  
SO Transplantation (1997), 64(6), 913-918  
CODEN: TRPLAU; ISSN: 0041-1337

L19 ANSWER 7 OF 23 CAPLUS COPYRIGHT 1998 ACS  
AU Horigome, Atsushi; Hirano, Toshihiko; Oka, Kitao; Takeuchi,  
Hironori; Sakurai, Etsuo; Kozaki, Koichi; Matsuno, Naoto;  
Nagao,  
Takeshi; Kozaki, Masami  
TI Glucocorticoids and cyclosporine induce apoptosis in  
mitogen-activated human peripheral mononuclear cells  
SO Immunopharmacology (1997), 37(1), 87-94  
CODEN: IMMUPD; ISSN: 0162-3109

L19 ANSWER 8 OF 23 CAPLUS COPYRIGHT 1998 ACS  
IN Baker, Robert K.; Bao, Jianming; Kayser, Frank; Parsons,  
William H.;  
Rupprecht, Kathleen M.  
TI Triterpene derivatives with \*\*\*immunosuppressant\*\*\*  
activity,

SO their preparation, and compositions containing them  
PCT Int. Appl., 121 pp.  
CODEN: PIXXD2

L19 ANSWER 9 OF 23 CAPLUS COPYRIGHT 1998 ACS  
AU Chan, Christopher; Maurer, Janet; Cardella, Carl; Cattran,  
Dan; Pei,  
York  
TI A randomized controlled trial of verapamil on cyclosporine  
nephrotoxicity in heart and lung transplant recipients  
SO Transplantation (1997), 63(10), 1435-1440  
CODEN: TRPLAU; ISSN: 0041-1337

L19 ANSWER 10 OF 23 CAPLUS COPYRIGHT 1998 ACS  
AU Murphy, Brendan G.  
TI Lipoprotein (a) and the kidney  
SO Nephrology (1997), 3(2), 139-142  
CODEN: NEPHF2; ISSN: 1320-5358

L19 ANSWER 11 OF 23 CAPLUS COPYRIGHT 1998 ACS  
AU Schulze-Lohoff, E.; Ogilvie, A.; Sterzel, R. B.  
TI Extracellular nucleotides as signaling molecules for renal  
mesangial  
cells  
SO J. Auton. Pharmacol. (1996), 16(6), 381-384  
CODEN: JAPHDU; ISSN: 0144-1795

L19 ANSWER 12 OF 23 CAPLUS COPYRIGHT 1998 ACS  
AU De Lima, Jose J. G.; Maranhao, Raul C.; da Conceicao, Maria;  
Latriilha, M.; Diamant, Jayme; Romao, Joao Egidio; Krieger,  
Eduardo  
M.; Pileggi, Fulvio  
TI Early elevation of lipoprotein(a) levels in chronic renal  
insufficiency  
SO Renal Failure (1997), 19(1), 145-154  
CODEN: REFAE8; ISSN: 0886-022X

L19 ANSWER 13 OF 23 CAPLUS COPYRIGHT 1998 ACS  
AU Faedda, Rossana; Pirisi, Mario; Satta, Andrea; Bosincu,  
Luisanna;  
Baroli, Ettore  
TI \*\*\*Immunosuppressive\*\*\* treatment of Berger's disease  
SO Clin. Pharmacol. Ther. (St. Louis) (1996), 60(5), 561-567  
CODEN: CLPTAT; ISSN: 0009-9236

L19 ANSWER 14 OF 23 CAPLUS COPYRIGHT 1998 ACS  
AU Bonnin, Maria R.; Gonzalez, Maria T.; Grino, Jose M.;  
Cruzado, Jose  
M.; Martinez, Jose M.; Navarro, Miguel A.  
TI Evolution of circulating C-terminal propeptide of type I  
procollagen  
in patients with \*\*\*chronic\*\*\* \*\*\*renal\*\*\*  
\*\*\*failure\*\*\*  
pre and post renal transplantation  
SO Eur. J. Clin. Chem. Clin. Biochem. (1996), 34(11), 897-900  
CODEN: EJCBE0; ISSN: 0939-4974

L19 ANSWER 15 OF 23 CAPLUS COPYRIGHT 1998 ACS  
AU Elisaf, M.; Mikhailidis, D. P.; Siamopoulos, K. C.  
TI Dyslipidemia in patients with renal diseases  
SO J. Drug Dev. Clin. Pract. (1996), 7(4), 331-48  
CODEN: JDCPFC; ISSN: 1357-9215

L19 ANSWER 16 OF 23 CAPLUS COPYRIGHT 1998 ACS  
AU Stenvinkel, Peter; Berglund, Lars  
TI Lipoprotein(a) in chronic renal disease  
SO Miner. Electrolyte Metab. (1995), Volume Date 1996, 22(1-3),  
16-21  
CODEN: MELMDI; ISSN: 0378-0392

L19 ANSWER 17 OF 23 CAPLUS COPYRIGHT 1998 ACS  
AU Kuroda, Minpei; Mimaki, Yoshihiro; Sashida, Yutaka; Hirano,  
Toshihiko; Oka, Kitaro; Dobashi, Akira  
TI A novel 16,23-epoxy-5.beta.-cholestane glycoside with potent  
inhibitory activity on proliferation of human peripheral  
blood  
lymphocytes from Ornithogalum saundersiae bulbs  
SO Chem. Pharm. Bull. (1995), 43(7), 1257-9  
CODEN: CPBTAL; ISSN: 0009-2363

L19 ANSWER 18 OF 23 CAPLUS COPYRIGHT 1998 ACS  
AU Agmon, Yoram; Brezis, Mayer  
TI Effects of nonsteroidal \*\*\*anti\*\*\* - \*\*\*inflammatory\*\*\*  
drugs  
upon intrarenal blood flow: selective medullary  
hypoperfusion  
SO Exp. Nephrol. (1993), 1(6), 357-63  
CODEN: EXNEEG; ISSN: 1018-7782

L19 ANSWER 19 OF 23 CAPLUS COPYRIGHT 1998 ACS  
AU Kinlen, L. J.  
TI \*\*\*Immunosuppression\*\*\* and cancer  
SO IARC Sci. Publ. (1992), 116(Mech. Carcinog. Risk Identif.),  
237-53  
CODEN: IARCCD; ISSN: 0300-5038

L19 ANSWER 20 OF 23 CAPLUS COPYRIGHT 1998 ACS  
AU Ismail, F. A.; Khalafallah, N.; Khalil, Said A.  
TI Disease and drug-induced changes in naproxen binding to  
plasma  
SO Drug Dev. Ind. Pharm. (1989), 15(1), 147-64  
CODEN: DDIPD8; ISSN: 0363-9045

L19 ANSWER 21 OF 23 CAPLUS COPYRIGHT 1998 ACS  
AU Langhoff, E.; Ladefoged, J.; Odum, N.  
TI Effect of interleukin-2 and methylprednisolone on in vitro  
transformation of uremic lymphocytes  
SO Int. Arch. Allergy Appl. Immunol. (1986), 81(1), 5-11  
CODEN: IAAAAM; ISSN: 0020-5915

L19 ANSWER 22 OF 23 CAPLUS COPYRIGHT 1998 ACS  
AU Tvedegaard, Erling; Szpirt, Wladimir; Nielsen, Maja  
TI Effect of \*\*\*chronic\*\*\* \*\*\*renal\*\*\* \*\*\*failure\*\*\*  
and  
methylprednisolone treatment on the uptake of labeled plasma  
cholesterol into the aorta of normocholesterolemic rabbits  
SO Atherosclerosis (Shannon, Irel.) (1983), 47(2), 199-209  
CODEN: ATHSBL; ISSN: 0021-9150

L19 ANSWER 23 OF 23 CAPLUS COPYRIGHT 1998 ACS  
AU Schusziarra, V.; Ziekursch, V.; Schlamp, R.; Siemenssen, H.

C.  
TI Pharmacokinetics of azathioprine under hemodialysis  
SO Int. J. Clin. Pharmacol. Biopharm. (1976), 14(4), 298-302  
CODEN: IJCBDX

=> log y

COST IN U.S. DOLLARS  
TOTAL

SINCE FILE

SESSION  
FULL ESTIMATED COST

ENTRY

65.11 44.31

STN INTERNATIONAL LOGOFF AT 17:31:11 ON 15 AUG 1998